A STUDY OF ATRIAL FIBRILLATION-CLINICAL, TRANSTHORACIC ECHOCARDIOGRAPHY AND TRANSESOPHAGEAL ECHOCARDIOGRAPHY CORRELATION

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CERTIFICATE

This is to certify that this dissertation titled *"**STUDY OF ATRIAL FIBRILLATION – CLINICAL, TRANSTHORACIC ECHOCARDIOGRAPHY AND TRANSESOPHAGEAL ECHOCARDIOGRAPHY CORREALATION"*** submitted by **DR. B. PRAKASH SHANKAR** to the faculty of General Medicine, The Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of MD Degree Branch I (General Medicine) is a bonafide research work carried out by him under our direct supervision and guidance.

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DECLARATION

I, Dr. **B. PRAKASH SHANKAR ,** *solemnly declare that the dissertation "**STUDY OF ATRIAL FIBRILLATION – CLINICAL, TRANSTHORACIC ECHOCARDIOGRAPHY AND TRANSESOPHAGEAL ECHOCARDIOGRAPHY CORREALATION"** has been prepared by me.*

This is submitted to the Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the regulations for the award of MD Degree Branch I (General Medicine)

Place : Madurai

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CONTENTS

Introduction

Atrial fibrillation is the most common sustained cardiac arrhythmia[1] and represents the most common clinically significant cardiac arrhythmia[2].

Atrial fibrillation is characterized by uncoordinated supraventricular (Atrial) activation associated with an irregular rapid ventricular response. Consequently, this irregularly irregular rhythm leads to deterioration of normal atrial mechanical function. Although the prevalence of atrial fibrillation remains under one percent in general population for those who are less than 60 years old, it is estimated to affect 6 to 10 percent of people above 80 years of age[3]. It has been clearly shown that the presence of atrial fibrillation may convey significant morbidity and mortality[4].

Atrial fibrillation is the most common arrhythmia that requires treatment and it crosses the path of almost all clinicians. Although the majority of patients with atrial fibrillation are relatively asymptomatic, patients can have profoundly limiting symptoms due to rapid or slow basal ventricular rates, a rapid cardio accelerator response to exercise, beat to beat irregularity with associated palpitations, and the loss of atrial systolic contribution to ventricular filling lead to diminished cardiac output. In addition, there is a propensity for left atrial thrombus with subsequent morbidity in the form of embolic stroke. For these reasons there

has been a strong impetus to improve our knowledge and understanding of atrial fibrillation and its response to therapeutic interventions[5].

Underlying causes of atrial fibrillation are structural heart diseases, and these compose most of the atrial fibrillation population. The most common cardiovascular pathologies associated with presence of atrial fibrillation in general population are valvular (mainly mitral) heart diseases, hypertension, coronary artery disease, congestive heart failure[6].

One population study has suggested that atrial fibrillation associated with different underlying structural abnormalities has a different age of first clinical appearance. Atrial fibrillation secondary to the valvular heart disease manifests approximately 15 years earlier than atrial fibrillation associated with hypertension or coronary artery disease[7].

HYPOTHESIS

Because of the poor understanding of the mechanism of atrial fibrillation, it routinely is classified as a single arrhythmia, but this is probably not the case, as many substrates and mechanisms may be responsible for a common ECG manifestation of atrial fibrillation. Just in the past decade, with the advancement of the technology used for atrial fibrillation mapping, understanding the pathophysiology of atrial fibrillation has grown immensely.

From the multiple wavelet theory that was first introduced by Moe and later confirmed through mapping by Allersie et al to the computer simulations of spiral waves and high frequency periodic sources seen with optical mapping, which have led to the development of mother rotor theory, controversy remains as to the pathophysiology of atrial fibrillation[8].

Atrial fibrillation carries a substantial risk of thromboembolism that also increases precipitously in the elderly (4 to 5 folds). Clinical risk factors have been identified from the stroke prevention in atrial fibrillation (SPAF TRIAL) trial. These include

1. Previous thromboembolism
2. Hypertension
3. Poor LV function, and
4. Age >75 years

Echocardiography has been helpful in further defining risks. Transthoracic echo cardiographic risk factors that were identified in SPAF trial include

1. LA size
2. LV dysfunction

In fact echo variables altered thromboembolic risk in 18% of the entire cohort study and in 38% of those with out any clinical risk factors[9].

Transesophageal echocardiography (TEE) offers unique imaging resolution of the left atrium and its appendages and is an excellent tool for detecting embolic sources. In SPAF trial 41% of patients underwent TEE examination. Transesophageal echocardiographic features that were independently associated with increased thromboembolic risk in patients taking aspirin were, appendage thrombi, dense spontaneous echo contrast, left atrial appendage peak flow velocities <20 cm\sec, and complex aortic plaque[10].

This study was done to evaluate the common causes of atrial fibrillation in our population and evaluate them clinically and by means of Transthoracic and Transesophageal echocardiography.

Review of literature

Perhaps the earliest description of atrial fibrillation is in the Yellow emperor's classic of internal medicine (Huang ti neiching suken). The legendary emperor physician is believed to have ruled China between 1696 and 2598 B.C. The poor prognosis associated with chaotic irregularity of the pulse was clearly acknowledged by most of the ancient physicians but in recorded history, William Harvey in 1628 was probably the first to describe "fibrillation of auricles" in animals[1].

In clinical practice, with the aid of Lannec's recently invented stethoscope, Robert Adams reported in 1827 the association of irregular pulses with mitral stenosis in 1863; Eitiene Marey published a pulse tracing with mitral stenosis. Other early descriptions of atrial fibrillation and its importance were published early in the century by Sir James Mackenzie and Heinrich Hering. The discovery of therapeutic properties of digitalis leaf (Digitalis purpurea) in 1785 by William Withering brought some relief to patients with severe heart failure. It is interesting to note that Withering recorded a patient who had a weak, irregular pulse that became "more full and regular" after five draughts containing Fol digital purp oz intravenous.

The main diagnostic breakthrough was the invention of the electrocardiography by William Einthoven in 1900. Sir Thomas Lewis at University college hospital was the first to record an electrocardiogram in a patient with atrial fibrillation.

History of atrial fibrillation[1]

1827	Adams, probably the first to recognize the condition clinically, but as a sign of mitral stenosis.
1839	Hope identified irregular pulse in association with mitral stenosis, exercise worsened the total irregularity, where as it abolished an intermittent pulse.
1863	Marey published a pulse tracing of atrial fibrillation from a patient with Mitral stenosis
1874	Vulpian, observed atrial fibrillation In vivo.
1894	Engelmann reported atrial fibrillation Caused by multiple foci in atria
1900	Einthoven invented ECG.
1909	Lewis recorded atrial fibrillation with Electrocardiography and studied mechanisms of conduction. Winterburg and Rothenburg identified “arrhythmia perpetua” and “fibrillation of auricles”.

1935	Bouilland found that digitalis reduced the ventricular rate dramatically even though irregularity of pulse persisted.
1969	Lown recommended cardio version of atrial fibrillation.

Epidemiology

Atrial fibrillation is common in the community, affecting up to 5% of the people aged 75 years. It is a major reason for emergency admissions and cause of cardiovascular deaths. Thus most clinicians in hospital and general practice will participate in managing such patients. As the prevalence of the condition increases with age, atrial fibrillation will become increasingly common in the increasingly aging population.

Epidemiological studies have shown that atrial fibrillation is fairly uncommon in people aged less than 50 years but is found in 0.5% of population aged 50 to 59 years, increasing to 8.8% at age 80 to 89 years. Furthermore the arrhythmia may be either chronic or paroxysmal. In the Framingham study, hypertension, cardiac failure and rheumatic heart disease were the commonest precursors of atrial fibrillation. Commonest causes in western countries include coronary artery disease, hypertension and rheumatic and non rheumatic valvular heart diseases.

In contrast, in developing countries Rheumatic heart disease is by far the most common cause of atrial fibrillation. The prevalence of Rheumatic heart disease in India is about 3.2% while study undertaken by CMC Vellore Hospital in the population living in and around Vellore has shown a prevalence rate of 0.69%.

From the available data it could be said that Rheumatic heart disease constitutes a very important etiology for atrial fibrillation.

Importance

Because of serious implications of atrial fibrillation, clinicians in all specialties as well as hospitals and primary health care nurses must be trained adequately in its detection and management. The sudden onset of fast atrial fibrillation may precipitate overt heart failure, particularly if left ventricular function is already compromised by the co existing heart diseases, such as valvular or ischemic heart disease. Less dramatic presentation of atrial fibrillation includes palpitation, dyspnea, angina, general fatigue and lethargy. Symptoms may be more pronounced on exercise, with a greatly limited exercise tolerance.

More important is however, the finding that non rheumatic atrial fibrillation increases the risk of stroke by a factor of five. The risk of stroke in atrial fibrillation is about 5% per year and epidemiological studies suggest that the risk increases with age, blood pressure, and other evidence of heart diseases. Atrial fibrillation may also increase the risk of recurrent stroke.

Definition

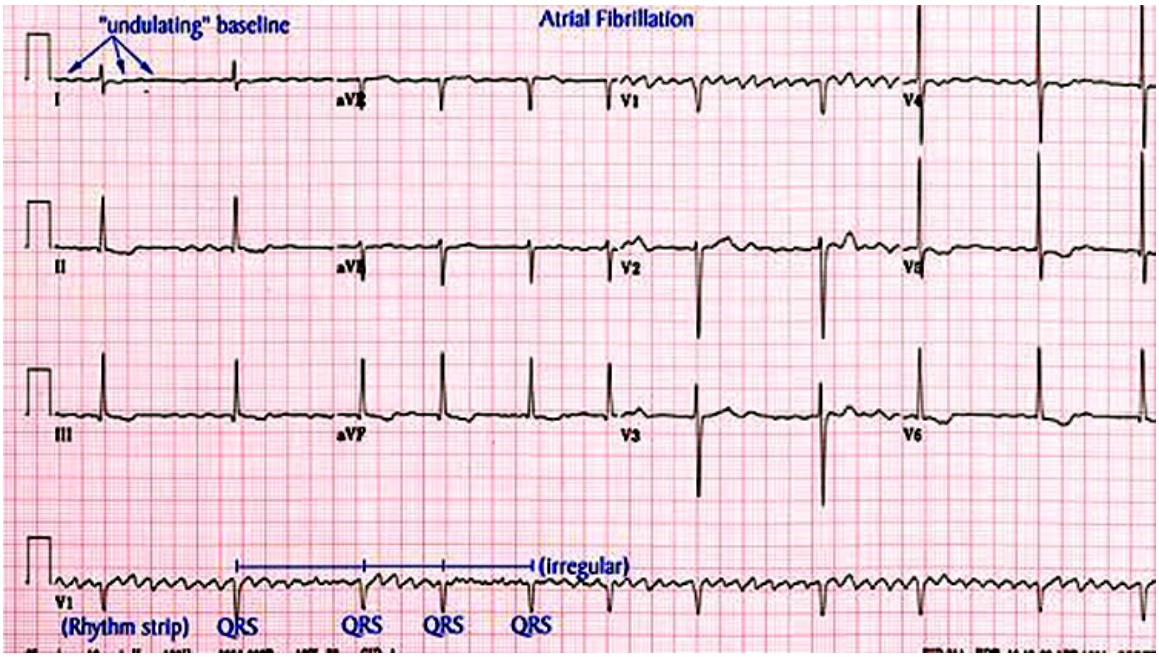
Atrial fibrillation is a supraventricular tachyarrhythmia characterized by uncoordinated atrial activation with consequent deterioration of atrial mechanical function.[11, 12]

On the electrocardiogram atrial fibrillation is described by the replacement of consistent p waves by rapid oscillations with an irregular, frequently rapid ventricular response when atrioventricular conduction is intact. The ventricular response to atrial fibrillation depends on electrophysiological properties of the

A-V node, the level of vagal and sympathetic tone, and the actions of drugs. Regular R-R intervals are possible in the presence of A-V block or interference due to ventricular or junctional tachycardia. In the patients with ectopic pace makers, diagnosis of atrial fibrillation may require temporary inhibition of the pace maker to expose atrial fibrillatory activity. A rapid irregular, sustained, wide QRS complex tachycardia strongly suggests atrial fibrillation with conduction over an accessory pathway or atrial fibrillation with underlying bundle branch block. Extremely rapid rates (over 200 bpm) suggest the presence of an accessory pathway[12].

Figure 1.

ECG of Atrial fibrillation



Diagnosis and screening for atrial fibrillation

Electrocardiogram and its limitations

The resting electrocardiogram remains the main stay diagnostic tool for diagnosis of atrial fibrillation. ECG is easy to perform and inexpensive, however the paroxysmal and frequently asymptomatic nature of atrial fibrillation in most patients limits the value of usual 12 lead ECG as a screening test of atrial fibrillation. Negative single surface ECG therefore has limited sensitivity, which may be improved by ECG monitoring (Holter loop recorder). In patients having pacemaker implanted, there is a unique possibility of monitoring the intra atrial electro cardiograms. It may be difficult however to extrapolate the data obtained from the devices with an atrial electrode, because many of the indications for implantation of a device are associated with increased incidence of atrial fibrillation. Also existence of atrial electrode itself may increase the risk of the chance of having atrial fibrillation in the future[13, 14].

Classification

A variety of the nomenclature terms have previously been used to describe atrial fibrillation, including “lone atrial fibrillation”, idiopathic atrial fibrillation” and non valvular atrial fibrillation. The most recently executive summary endorsed by the American College of Cardiology, the American Heart Association, the European Society of Cardiology, and the North American Society

of Pacing and Electrophysiology had classified atrial fibrillation as, paroxysmal, persistent and permanent.[14,15,16] These terms are defined below:

1. Paroxysmal: recurrent, intermittent atrial fibrillation that previously terminated with out specific therapy. Paroxysmal atrial fibrillation is self limited.

2. Persistent: recurrent, sustained atrial fibrillation that was previously terminated by therapeutic intervention. Persistent atrial fibrillation may be the first presentation, a culmination of recurrent episodes of paroxysmal atrial fibrillation or long lasting atrial fibrillation (greater than one year). Persistent atrial fibrillation is not self limited, but may be converted to sinus rhythm by medical or electrical interaction.

3. Permanent: continuous atrial fibrillation which cannot be converted to normal sinus rhythm by pharmacological or electrical techniques.

In the Cox classification system, if atrial fibrillation is present all the time, it is defined as continuous atrial fibrillation and if the atrial fibrillation is not present all the time, it is defined as intermittent atrial fibrillation. This important distinction is directly linked to the therapeutic decision making. Simple pulmonary vein encirclement may provide an adequate cure for those patients with intermittent atrial fibrillation, since in this subset of patients; pulmonary veins usually provide the necessary aberrant electrical signals. However for patients

with continuous atrial fibrillation simple pulmonary vein isolation procedure is not usually adequate therapy.

Factors that contribute to Atrial fibrillation

Potentially reversible causes of AF

Electrolyte abnormalities

Intoxicants: alcohol, carbon monoxide

Cardiothoracic surgery

Electrocution

Pulmonary embolism

Other pulmonary diseases

Hyperthyroidism

Cardiovascular diseases associated with AF

Systemic hypertension

Congestive heart failure

Valvular heart disease

Inflammatory atrial disease: myocarditis, pericarditis

Infiltrative atrial disease: amyloidosis, age-related fibrotic changes

Coronary artery disease

Primary or metastatic disease involving the atrial wall

Congenital heart disease: atrial septal defect, Ebstein's anomaly,

Postsurgical repair

Neurogenic and autonomically mediated causes of AF

Heightened vagal tone

Heightened adrenergic tone, resulting from anxiety,

Pheochromocytoma, exertion

Subarachnoid hemorrhage

Basic Mechanisms and Genetics

Because of the poor understanding of the mechanisms of atrial fibrillation it is routinely classified as a single arrhythmia, but it probably is not the case as many substrates and mechanisms may be responsible for a common ECG manifestation of atrial fibrillation. Just in the past decade with the advent of technology used for atrial fibrillation mapping, understanding of pathophysiology of atrial fibrillation has grown immensely. From the Multiple wavelet theory that was first introduced by Moe et al[18] and later confirmed through mapping by Allersie et al[19], to computer simulations of spiral waves and high frequency periodic sources seen with optical mapping which have led to the development of the "Mother rotor theory", controversy remains as to the pathophysiology of atrial fibrillation[8].

Figure 2

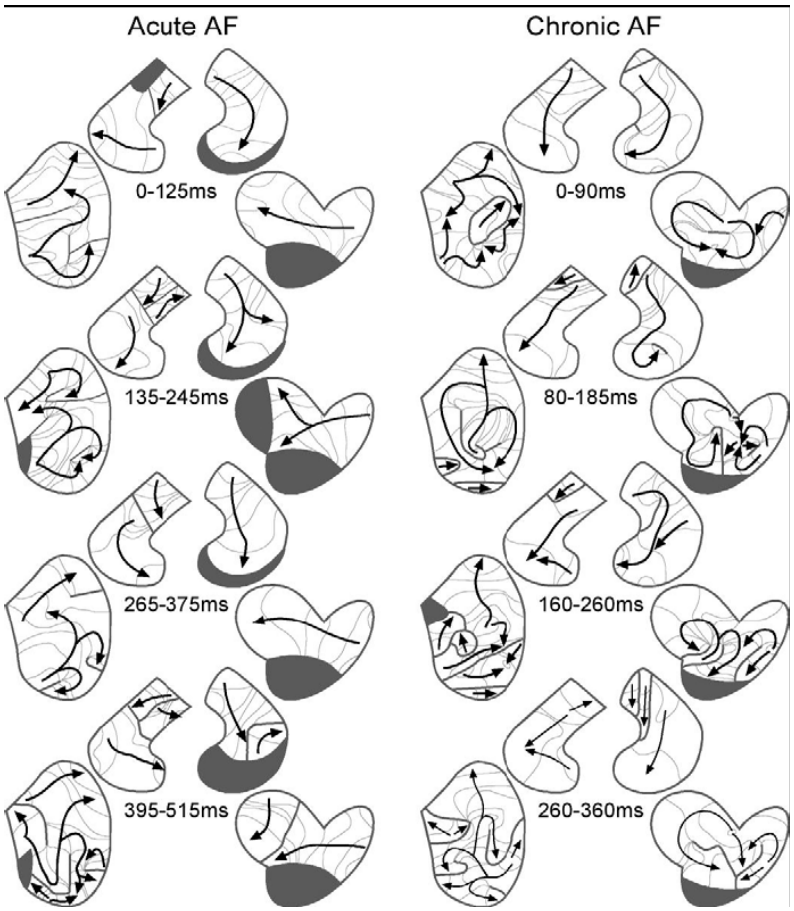


Figure 2 Activation maps recorded during acute and chronic AF with high-density epicardial plaque electrodes. The gray regions indicate areas that were not in contact with the atria. Both the acute and chronic AF maps show that the AF in this model is composed of multiple wavelets.

Specific mechanisms

Multiple wavelets Theory

The idea that, electrical impulses could have re-entrant characteristics was first proposed by Mayer in two papers that were published in 1906 and in 1908. In these two studies rings of tissue from jelly fish were used for study.

Lewis suggested that atrial fibrillation was composed of a single re -entrant circuit with varying durations. The mechanism of atrial fibrillation was considered to be due to either circuit movement of re -entry or a variation of this theory, until Moe proposed the theory that atrial fibrillation was caused by multiple, random re-entrant wavelets. The more wavelets that existed, the more likely the fibrillation would continue. This hypothesis was regarded widely as the mechanism of atrial fibrillation, and it gained support when Allersie et al showed that based on high density mapping of a canine heart during atrial fibrillation, its mechanism was multiple wavelets. They concluded that four to six wavelets are needed for continuation of atrial fibrillation[8].

Mother rotor theory:

A rotor has been defined as a stable rotating pattern of reaction and diffusion that surrounds a pivot point, also known as a phase singularity. A curved wave front radiates from the rotor into the surrounding tissues[20].

Optical movies were examined and the isochronal maps constructed and it was determined that these high frequency sources were a vortex rotating clockwise. This rotor lasted for the entire episode of atrial fibrillation. Because the frequency of the rotor was the highest frequency of all recorded sites, it was determined that the rotor was driving the atrial fibrillation. Hence a mother rotor was defined as a stable, high frequency source that appears to be driving the atrial fibrillation. All of these high frequency sources were found in the left atrium suggesting that the left atrial activity was driving the atrial fibrillation.

Pulmonary veins and focal source

Until recently it was believed widely that the multiple re -entrant wave fronts associated with atrial fibrillation did not have a single point of origin, but recent studies have demonstrated that paroxysmal atrial fibrillation can have a focal source and the pulmonary veins plays a major role in the origin of the focal source[21]. Haissagurre et al showed that 94% of the single point origin was identified as originating from the pulmonary veins with the earliest activation occurring 2 to 4 cm with in pulmonary veins.

The mechanism of atrial fibrillation remains controversial as support still exists for multiple wavelets, “mother rotor” and focal sources. These mechanisms need not be mutually exclusive. For example, the mother rotor hypothesis may not be distinct from the focal atrial fibrillation, if the rotor is of small size (i.e. micro-entry). It is likely that there is not one mechanism for all atrial fibrillation, but that

there are substrate specific mechanisms and that atrial fibrillation may be comprised of several different mechanisms.

Genetic studies showed that mutations in GJA5 genes may predispose patients to idiopathic atrial fibrillation by impairing gap junction assembly or electrical coupling. The cardiac gap junction protein connexin-40 is expressed selectively in atrial myocytes and mediates the coordinated electrical activation of the atria. Hence the tissue specific mutations in GJA5, the gene coding connexin-40, may predispose the atria to fibrillation[22].

Clinical evaluation

The initial evaluation of a patient with atrial fibrillation begins with a thorough history focused on identifying precipitants, defining associated cardiac and extra cardiac factors, and characterizing the pattern of arrhythmia (e.g. symptoms, duration, paroxysmal vs. persistent, first episode vs. recurrent etc). Physical examination typically reveals an irregularly irregular pulse, irregular jugular venous pulsations with absent “A waves” and variations in the intensity of the first heart sound[23]. Associated valvular disease, primary or secondary (i.e. tachycardia induced) cardiomyopathies, or heart failure may be identified. The definite diagnosis of atrial fibrillation requires at least one ECG lead documenting the arrhythmia from a rhythm strip, standard 12 lead ECG, holter monitoring, or transtelephonic or telemetric recording. If episodes are infrequent an external or internally implanted event recorder may allow the patient to transmit the stored ECG to a recording facility when symptoms occur[24].

RISK OF EMBOLISATION

In addition to hemodynamic alterations, the risk of systemic emboli, probably arising in the left atrial cavity or appendage as a result of circulatory stasis, is an important consideration.

In United States Non valvular atrial fibrillation is the most common cardiac disease associated with cerebral embolism. In fact almost half of cardiogenic emboli in United States occur in patients with non valvular atrial fibrillation. The risk of stroke in patients with non valvular atrial fibrillation is five to six times greater than in controls without atrial fibrillation. Over all 20 to 25 percent of ischemic strokes are due to cardiogenic emboli[25].

Table 1[26]

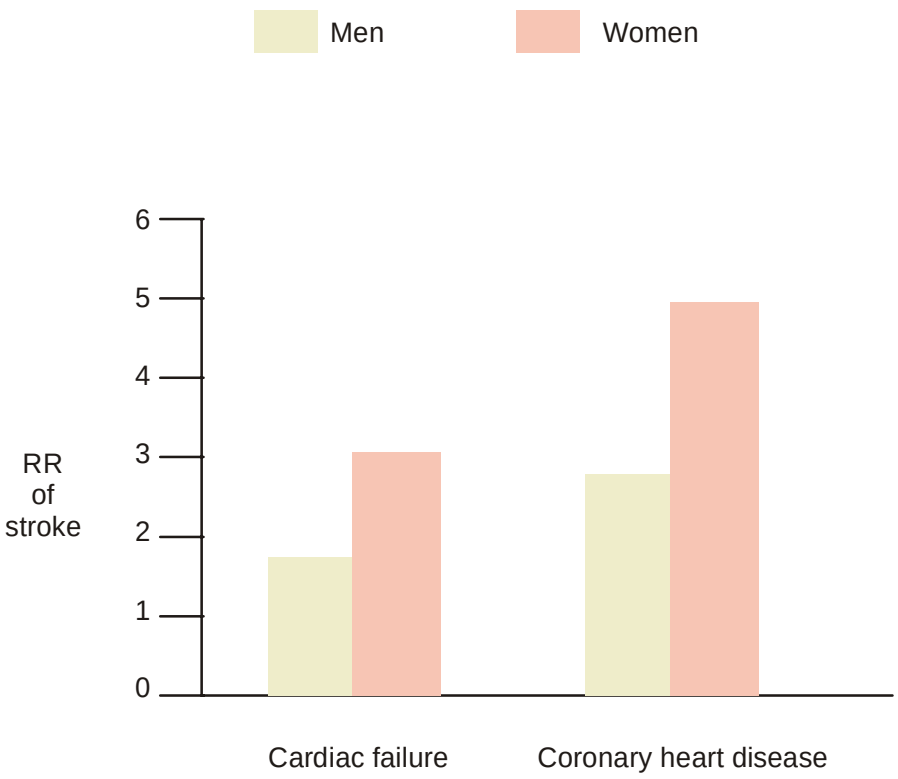
Risk Factors (Control Groups)	Relative Risk
Previous stroke or TIA	2.5
History of hypertension	1.6
Congestive heart failure	1.4
Advanced age (continuous, per decade)	1.4
Diabetes mellitus	1.7
Coronary artery disease	1.5

From ACC/AHA/ESC Guidelines for the Management of Patients with Atrial Fibrillation, 2002.

Table shows the risk factors for thromboembolism in patients with atrial fibrillation.

Figure 3

Age-Adjusted Relative Risk of Stroke in Atrial Fibrillation With CHF or CHD



Risk factors that predict stroke in patients with non valvular atrial fibrillation include a history of previous stroke or transient ischemic attack (relative risk 2.5), diabetes (relative risk 1.7) history of hypertension (relative risk 1.6) and increasing age (relative risk 1.4 for each decade). Patients with any of these risk factors have an annual stroke risk of at least 4 percent if untreated. Patients whose only stroke risk factor is congestive heart failure or coronary artery disease have stroke rates approximately three times higher than do patients with out any risk factors. Left ventricular dysfunction and a left atrial size greater than 2.5 cm\m^2 on echocardiographic examination are associated with thromboembolism. Patients younger than 60 to 65 years of age who have a normal echocardiography and no risk factors have an extremely low risk of stroke (1 percent per year). Therefore the risk of stroke in patients with lone atrial fibrillation in the absence of any structural heart disease or any of the risk factors discussed previously is quite low.

But these datas are from western part of the globe and unfortunately we don't have sufficient data or clinical trials regarding atrial fibrillation in India, predominantly caused by valvular heart diseases particularly that of mitral valve. Patients with mitral stenosis and atrial fibrillation have a 4 to 6 percent incidence of embolism per year.

Table 2

Stroke and Atrial Fibrillation according to Age – Framingham Study

Age (yr)	% Strokes with AF	% Strokes Attributed to AF
50-59	6.5	1.5
60-69	8.5	2.8
70-79	18.8	9.9
80-89	30.7	23.5

The Framingham study in the table 2 clearly shows the increasing percentage of stroke with atrial fibrillation, with increasing age. But this study was conducted among the western population with predominantly non valvular atrial fibrillation. The percentage of stroke attributed to atrial fibrillation in the age group of 50-59 years is only 1.5% where as in the age group of 80-89 years it is about 30.7%.

Clinical guidelines

American college of cardiology, the American heart association and the European society of cardiology recently established joint guidelines for atrial fibrillation management.

When an episode of atrial fibrillation is first detected, it should be described according to whether it is symptomatic or not, or self limited or not. Atrial fibrillation is considered to be recurrent if 2 or more episodes have been documented. If atrial fibrillation terminates spontaneously it is considered to be “paroxysmal”. Non self terminating atrial fibrillation is considered to be persistent, regardless of whether cardio version is performed pharmacologically or electrically. If cardio version is not indicated or attempted and the patient remains in atrial fibrillation, the arrhythmia is designed “permanent”.

Table 3: Clinical classification of AF

First episode of AF

• Symptomatic or asymptomatic

• Self-limited or persistent

Recurrent AF 2 or more episodes of AF lasting > 30 s

Paroxysmal AF Recurrent AF that has ended spontaneously

Persistent AF AF that requires pharmacological therapy or electrical cardio version for termination; may be a first episode or recurrent AF

Permanent AF Long-standing AF (usually > 1 yr) in which cardio version has failed or has not been indicated

Investigations

Transthoracic echocardiography

Transthoracic echocardiography is performed commonly during the initial evaluation of atrial fibrillation, when it enables clinicians to screen for occult pericardial, myocardial and valvular diseases. This examination may offer clues to etiology of dysrrythmia and provide information that will alter the approach to therapy. Several studies have suggested that information acquired by transthoracic echocardiography can assist clinicians with the sometimes difficult decisions of initiating antithrombotic prophylaxis in patients with atrial fibrillation. Transthoracic echocardiography is limited, however by inadequate visualization of the left atrial appendage from where most cardio embolic events are believed to originate.[27, 28, 29]

Transthoracic echocardiography is really necessary for the initial evaluation and management in patients who have a first episode of atrial fibrillation.

Several are the disorders associated with atrial fibrillation; some of the most important are mitral stenosis, left ventricular hypertrophy, focal wall motion abnormalities suggestive of myocardial infarction, left ventricular dysfunction and others which are diagnosticated fast and precociously by echocardiography. The information on left ventricular systolic function helps to guide the choice of pharmacological therapy for ventricular rate control in chronic atrial fibrillation.[30]

Transesophageal echocardiography

Transesophageal echocardiography employs a miniature, high frequency ultrasound mounted on a steerable endoscope to visualize the heart from within the esophagus, eliminating sonographic interference from the lung and chest wall that limits standard transthoracic view.

Transesophageal echocardiography is safe in the hands of an experienced operator but mastery of the technique requires specialized training and performance of an adequate number of studies to ensure high quality imaging[31].

The left atrial appendage is an intricate, frequently multilobed muscular extension of the left atrium that lies in close proximity to the esophagus. Meticulous imaging of the left atrial appendage in several planes is essential because of the structure's complexity and anatomical variability among subjects. Veinot et al noted that 80% of specimens had multilobed appendages, with some demonstrating as many as four chambers[32]. Pathological and echocardiographic studies have implicated embolism of preexisting thrombus from left atrial appendage as a predominant etiology of the stroke in patients with atrial fibrillation.

Transesophageal echocardiography is superior to Transthoracic echocardiography in visualizing this structure[32], and identifying the left atrial thrombus, with sensitivity that approaches 100%.[33, 34]

Figure 4

Adipose tissue simulating thrombus in Transesophageal echocardiography.

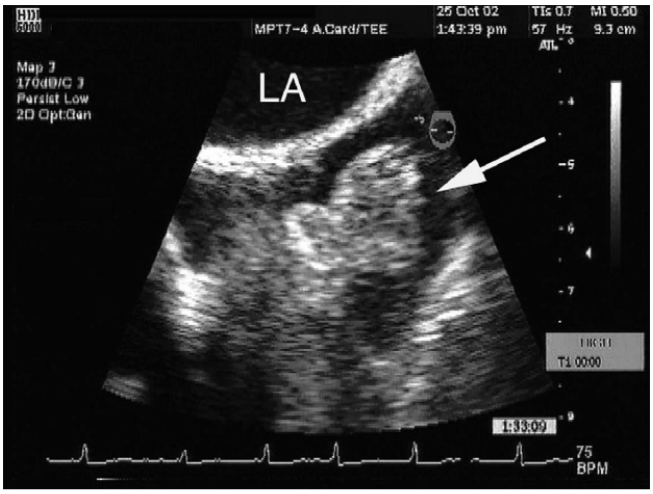
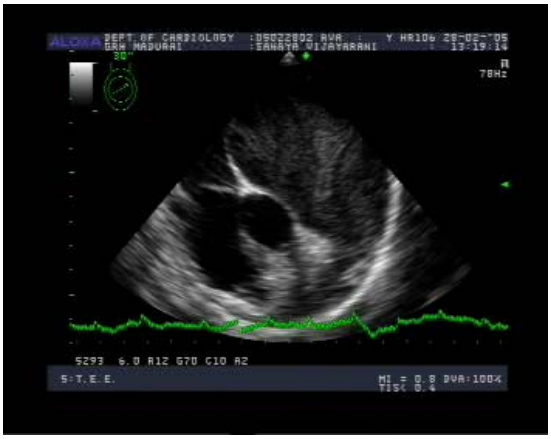


Fig. 2. Adipose tissue in the transverse sinus simulating a thrombus. This artifact is particularly common when there is a pericardial effusion. It can be differentiated from true thrombus by its location adjacent to the appendage, and Doppler will show no significant flow velocities in this structure, unlike the LAA.

Figure 5

Spontaneous Echo contrast in Transesophageal echocardiography.



Limitations of Transesophageal echocardiography[35]

1. Thrombus identification may be challenging even if the left atrial appendage is visualized adequately.
2. Several artifacts can result in misdiagnosis and lead to unnecessary delays in cardio version. E.g. prominent trabeculations, duplication artifacts, and adipose tissue with in the transverse sinuses.
3. Failure to adequately image the right atrial appendage, where the thrombi can form and clinically result in pulmonary embolism if left untreated.

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Figure 6

Left atrial clot in Transesophageal echocardiography.

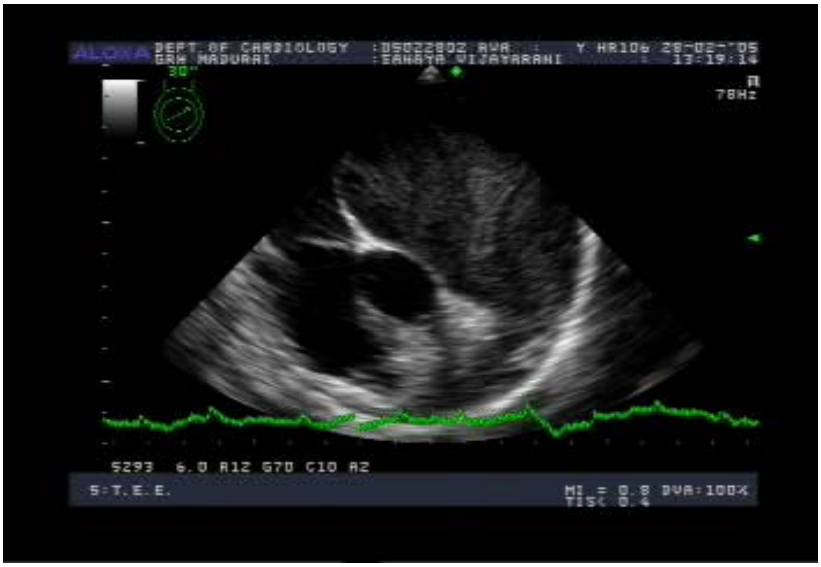
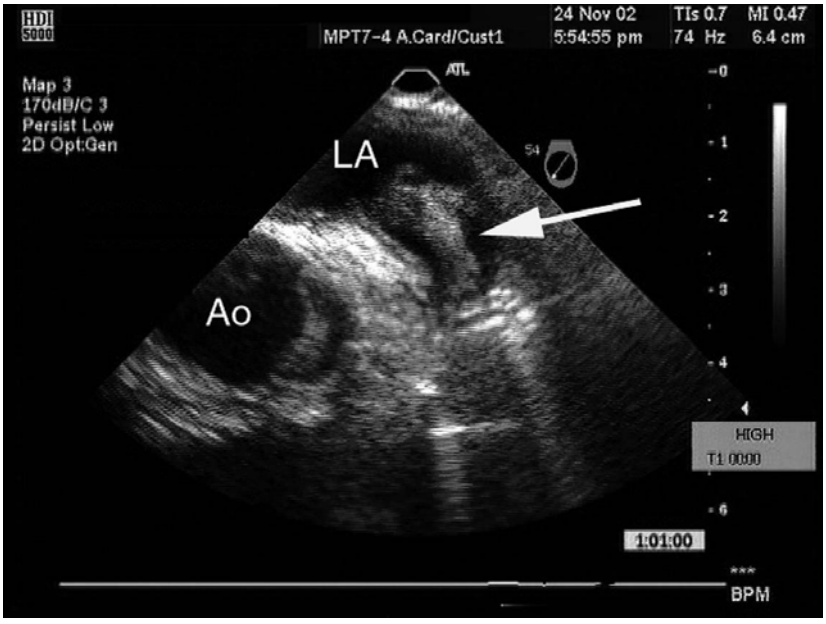


Figure 7

Left atrial appendage clot in Transesophageal echocardiography.



Correlation between Transthoracic and Transesophageal echo cardiography

A prospective study conducted by Khatouri et al showed that

Surgical findings in pure or very predominant mitral stenosis are more closely correlated with Transesophageal echocardiography which tends to overestimate the severity of anatomical lesions, particularly valvular calcifications. Both procedure can act in complement to each other with Transthoracic Echocardiography done first to evaluate the under lying cause of Atrial fibrillation and Transesophageal Echocardiography used to evaluate the risk of Left atrial thrombus formation. Transthoracic echocardiography is used to detect the underlying abnormality which predisposes to the atrial fibrillation and this may be followed by the Transesophageal echocardiography which helps to evaluate the risk of thrombus formation.

Left Atrial Appendage Flow Velocities:

In the normal state the left atrial appendage is relatively protected from thrombus formation by high velocity blood flow. Lower flow velocities are typical in atrial fibrillation however and have been associated with the presence of spontaneous echo contrast and clot formation.[36]

Left atrial appendage function can be assessed by pulse wave Doppler. In patients in sinus rhythm, the appendage contracts once per cardiac cycle and the flow velocities at the ostium demonstrate a biphasic pattern with peak velocities generally exceeding 40 cm\sec.

Figure 8

Decreased left atrial appendage flow velocity

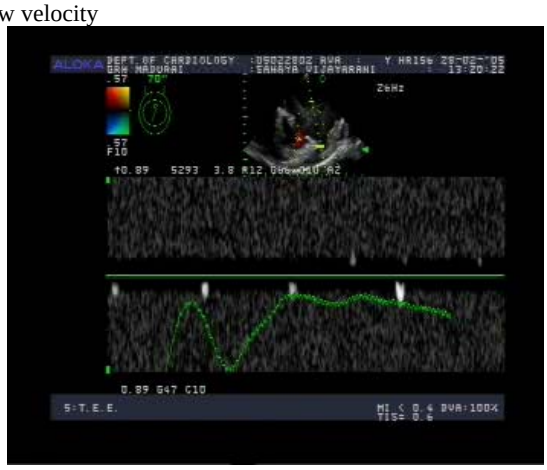
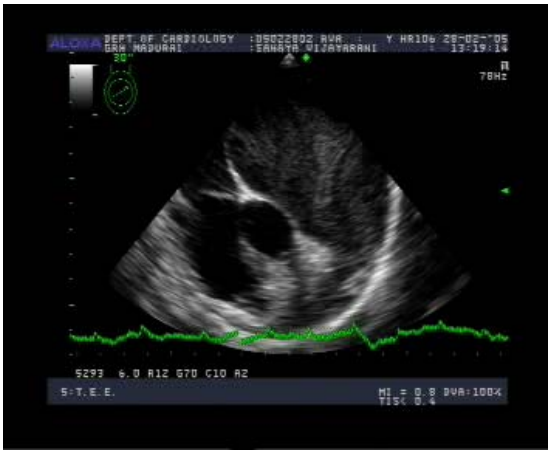


Figure 9

Spontaneous Echo contrast in Left Atrium

Spontaneous Echo Contrast (SEC)



Gracia Fernandez et al identified three distinct flow patterns in 39 consecutive patients, 18 of whom were in sinus rhythm.

Type 1 flow was characterized by distinct biphasic flow and was seen exclusively among patients in sinus rhythm.[37]

Type 2 flow is characterized by a rapid saw toothed pattern, appreciated in 38% of atrial fibrillation patients and correlated with larger left atrial appendage dimensions and spontaneous echo contrast.

In Type 3 flow there is no discernible waves were detected and the incidence of the thrombus was high.

A sub study of SPAF III demonstrated utility of left atrial appendage flow velocities to prospectively identify individuals at high risk. LAA peak flow velocities less than 20 cm\second were associated independently with an increased risk of thromboembolic events with a relative risk of 1.7.[38]

Spontaneous Echo Contrast (SEC)

Spontaneous echo contrast is identified commonly among patients with atrial fibrillation, and numerous investigators have been interested in the cause and prognostic significance of this finding.

The wispy, smoke like ECHO reflections observed are thought to be produced by back scatters from red cell aggregates at low flow rates and they are markers of

hematological stasis and a prothrombotic environment.[39, 40] Under normal conditions high flow velocities prevent erythrocytes from aggregating by maintaining shear stress. In atrial fibrillation the low flow state diminishes shear stress, leading to roleaux formation of red cells. Clinical, hematological and hemodynamic variables that have been associated independently with the presence of spontaneous echo contrast include age, fibrinogen levels, hematocrit, LAA velocities and atherosclerotic aortic plaque.[39, 40, 41]

In SPAF III study dense SEC was associated independently with increased thromboembolic risk with a relative risk of 3.7.

The presence of mitral regurgitation is negatively associated with SEC among patients with chronic atrial fibrillation, but its role in inhibiting thrombus formation and preventing stroke is less clear.

Because of the frequent association between SEC and cardio embolic events, a thorough investigation of LAA is warranted whenever dense SEC is identified. Even if thrombus is not found, these patients should be considered at high risk for embolic events and treated appropriately.

Distinguishing between dense contrast and true thrombus often can be difficult, as the two frequently coexist.

Despite the added prognostic information that can be acquired by Transesophageal echo cardiography, controversy exists as to the value of routine screening of all with atrial fibrillation for the purpose of risk stratification,

particularly those defined as low risk. The relatively low rate of thromboembolism in patients categorized as low risk based on clinical and standard echocardiographic parameters does not justify proceeding with routine TEE in this population. Individuals at moderate risk for both stroke and hemorrhagic complications with anticoagulation however may derive benefit from further risk stratification

Mitral valve area and thrombus formation

The incidence of thrombus in left atrium is significantly associated with mitral valve area in cases of mitral stenosis (a prospective study of left atrial spontaneous echo contrast) and also with the size of left atrium. But there are studies contradicting this aspect. In cases of non valvular atrial fibrillation left atrial size has no significant association with left atrial thrombus formation.

Table 4 : Determinants of the Echocardiographic Mitral Valve Score

Grade	Mobility	Subvalvular thickening	Thickening	Calcification
1	Highly mobile valve with only leaflet tips restricted	minimal thickening just below the mitral leaflets	leaflets near normal in thickness (4-5cm)	a single area of increased echo brightness
2	Leaflet mid and basal Portions have normal mobility	Thickening of chordal structures extending up to one third of the chordal length	Midleaflets normal, considerable thickening of margins	Scattered areas of brightness confined to leaflet margins
3	Valve continues to move forward in diastole, mainly from the base	Thickening extending to distal third of the chords	Thickening extending through the entire leaflet (5-8 cm)	Brightness extending in to the midportion of the leaflets
4	No or minimal forward movement of the leaflets in diastole	Extensive thickening and shortening of all chordal structures extending down to the papillary muscles	Considerable thickening of all leaflet tissue (>8-10 mm)	Extensive brightness throughout much of the leaflet tissue

Mitral valve severity score

Mitral valve severity score is used to assess the severity of mitral stenosis by grading the mitral valve leaflet mobility, subvalvular thickening, and thickening of valve leaflet and calcification of leaflets. When the score is >8 mitral stenosis is usually severe requiring valve replacement surgery. There are few studies showing positive association with the incidence of left atrial thrombus and the mitral valve severity score.

AIMS AND OBJECTIVES

1. **To study the clinical profile of all adult patients with atrial fibrillation admitted in Government Rajaji Hospital.**

2. **To evaluate the cause of atrial fibrillation in these patients.**

3. **To perform a comparative analysis of Transthoracic and Transesophageal echocardiography in atrial fibrillation.**

Materials and Methods

Type of study	:	Prospective analytical study
Setting	:	Department of Medicine, Government Rajaji Hospital, Madurai.
Collaborating Department	:	Department of Cardiology, Government Rajaji Hospital, Madurai.
Duration of study	:	August 2004 to June 2005
Ethical clearance	:	Ethical clearance was obtained.
Consent	:	Informed consent was obtained before taking up each case for study.

Inclusion criteria :

All patients above 18 years of age with atrial fibrillation in Surface electrocardiogram were included in the study.

The patients admitted in the medical and cardiology wards were taken for this study.

For the history of Rheumatic fever the past history with fever, migratory joint pain with no residual deformity were included.

To diagnose atrial fibrillation absent p waves, fibrillatory waves, irregularly irregular ventricular reponse in electrocardiography were taken as the evidence for diagnosis.

For evaluation regarding etiology the electrocardiograph, echocardiogram, chest X-ray were done in all cases.

Chronic obstructive pulmonary disease is diagnosed with Chest X-ray, electrocardiograph, echocardiogram, history of chronic cough and history of smoking.

Significant Q wave in ECG, Regional wall motion abnormality in echocardiography were taken as evidences for coronary artery disease.

Thyroid profile studies were done for ‘at risk’ cases only.

Diagnosis of hypertension was made with systolic BP $\geq$ 140 mmHG and /or Diastolic BP $\geq$ 90mmHG.

Exclusion criteria :

1. Patients with age less than 18 years.
2. Patients who were hemodynamically unstable.

Materials

152 patients with atrial fibrillation who were above 18 years of age and hemodynamically stable were included in the study.

Methods

152 patients with atrial fibrillation were studied and their selected clinical; socio demographic data were included in the proforma.

Patients age, sex, history of Coronary Artery Disease, Systemic Hypertension, Cardiomyopathies, Congenital Heart Disease, Thyroid dysfunctions, COPD, cerebrovascular accidents, transient ischemic attacks were taken in to account.

Clinical examination

1. Special attention for thyroid swelling,
2. Pulse rate,
3. Heart rate,
4. Pulse deficit,
5. Jugular venous pressure.

Laboratory data

1. 12 lead ECG with rhythm strip,
2. Transthoracic echocardiographic examination,

3. Transesophageal echocardiographic examination,
4. Thyroid function tests in appropriate patients,
5. Blood glucose levels-fasting and post prandial,
6. Chest X-Ray PA View.

Transthoracic echocardiography was done in all patients and the following parameters were assessed.

1. ejection fraction
2. left atrium size
3. left atrial clot
4. spontaneous echo contrast
5. mitral valve severity score
6. mitral valve area

All patients were analyzed with 2D ECHO, M MODE and Color Doppler to find out the structural heart disease like congenital heart diseases, coronary heart diseases, hypertensive heart disease, and dilated cardiomyopathies. Transthoracic echocardiographic assessment also included the search for the presence of left atrial thrombus, left atrial appendage thrombus, left atrial auto contrast.

Transesophageal echocardiography was done in all patients and following parameters were assessed.

1. left atrial appendage size
2. left atrial appendage flow velocity(filling and emptying)
3. left atrial thrombus
4. spontaneous echo contrast
5. clot size

Transesophageal echocardiography employs a miniature, high frequency ultrasound transducer mounted on a steerable endoscope to visualize the heart from with in the esophagus, eliminating sonographic interference from the lung and the chest wall that limits transthoracic view.

All patients underwent Transesophageal echocardiographic evaluation using ALOKA SSPPRO 2000 with multiplane TEE probe. Left atrial appendage emptying velocity is normally > 0.6 mt/sec. Left atrial dimension is normally 1.92 to 4 cm. Normal mitral valve orifice is 4-6 $cm^{2.}$

Limitations of the study

1. Patients with age less than 18 years were not included in the study due to difficulty encountered during Transesophageal echocardiographic study.
2. Atheromatous aorta which could produce thromboembolism in atrial fibrillation patients was not included in the study, because the association between the two is not well supported by studies.
3. Transesophageal echocardiography could not effectively document thrombus in right atrial appendage, so the study was limited to left atrium and its appendage.
4. Patients with hemodynamic instability were not included in the study so the association between the incidence of left atrial thrombus and the LV dysfunction could not be completely assessed.

Financial support – nil

Competing\conflicting interest- nil

Statistical analysis – Epidemiological Information Package – 2005 was used to calculate the frequencies, percentages, mean, standard deviations and p values.

A ‘p’ value less than 0.05 is taken to represent significant difference.

Results

Total number of patients included in our study was 152

Table **5: Baseline Characters**

Total Number of cases	152
Mean age in years (SD)	35.6±10
Males	66 (43.4%)
Females	86 (56.6%)
Rheumatic heart disease	134 (88%)
Hypertension	13
Thyrotoxicosis	4
Atrial Septal Defect	2
Dilated cardiomyopathy	2
Hypertrophic ObstuctiveCardioMyopathy	2
Coronary artery disease	2
Rheumatic heart disease	Number of patients
Mitral Stenosis	122
Mitral Regurgitation	78
Aortic Regurgitation	32
Aortic Stenosis	6

The mean age of our study population was 35.6± 10 years. Female patients were predominant in our study and 86 patients were female patients (57%). In our study majority of population had rheumatic heart disease. We divided the study population into rheumatic and non rheumatic group. 134 patients (88%) had rheumatic heart

disease and 18 patients (12%) belonged to non rheumatic group. Out of the non rheumatic group 6 patients had hypertension and 4 patients had Thyrotoxicosis, 2 patients had atrial septal defect, 2 patients had dilated cardiomyopathy, 2 patients had Hypertrophic obstructive cardiomyopathy, 2 patients had coronary artery disease and 7 patients had both rheumatic heart disease and hypertension. In rheumatic heart disease group 122 patients (91%) had mitral stenosis. 89 of them had severe mitral stenosis and 25 patients had moderate mitral stenosis and 8 patients had mild mitral stenosis. 78 patients had mitral regurgitation, 34 patients had aortic regurgitation, 6 patients had aortic stenosis.

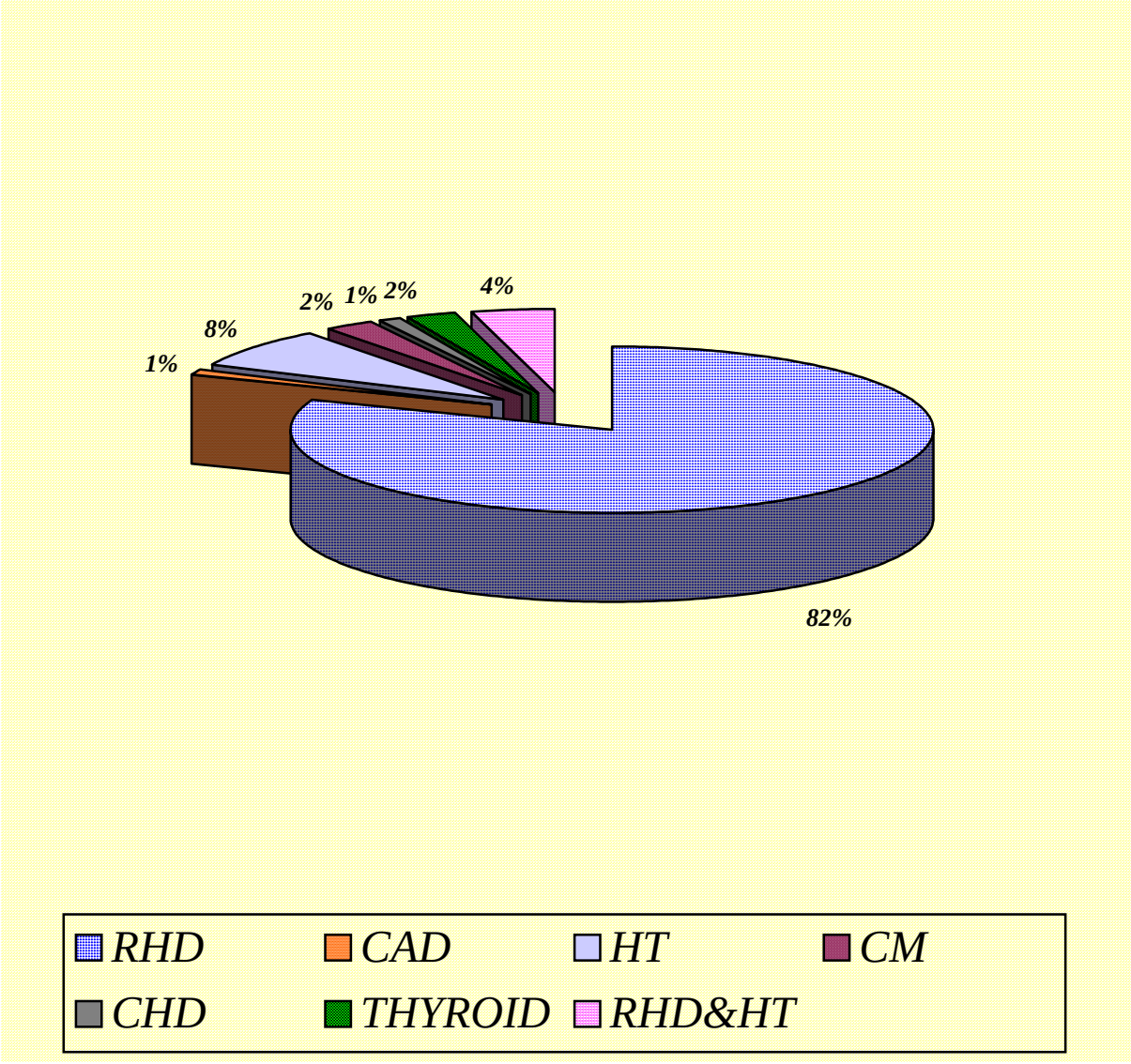
Table 6 : Profile of our study population

	Rheumatic Heart Disease		**Non rheumatic Heart Disease**	
	134(88%)		18(12%)	
	Thrombus	**No Thrombus**	**Thrombus**	**No Thrombus**
	74 (55%)	60 (45%)	4 (27%)	14 (73%)
AGE	34±7	34±10	40±6.9	50±8
MALE	38(51%)	20(32%)	2(60%)	6(58%)
FEMALE	36(49%)	42(68%)	2(40%)	8(42%)
Left Atrial area in cm^2	4.7±0.3	2.5±0.3 P=0.004	4.5±2.1	4±0.4 P=0.641
Left Atrial Appendage emptying Velocity in metre/sec.	0.165 ± 0.11	0.31±0.08 P=0.001	0.169±0.17	0.35±0.1 P=0.026

The study population was broadly divided into two groups namely those with rheumatic heart disease and those without rheumatic heart disease. About 134 patients had rheumatic heart disease (88%) out of which 74 patients (55%) had left atrial thrombus and 60 (45%) patients did not have thrombus. In those who had thrombus the mean size of left atrium was 4.7 cm and left atrial appendage emptying velocity was 0.17 m\s. In patients without thrombus the left atrial dimension was 2.5±0.3 cm, left atrial appendage flow emptying velocity was 0.36m\s.

Figure 10

Causes of Atrial Fibrillation



Causes of Atrial fibrillation in our study

Table 7: Causes of Atrial fibrillation in our study

Causes	**No.**	**%**
CAD	2	1.3
RHD	134	88.2
HT	13	8.6
CM	4	2.7
CHD	2	1.3
Thyroid	4	2.7

In this study group of atrial fibrillation, predominant cause was Rheumatic heart disease, constituting about 134 cases (88%). Other causes grouped together constituted only 18 cases (12%). There was a statistically significant difference of 0.001. Among the rheumatic heart disease group, mitral stenosis was the predominant cause, with 122 patients diagnosed to have mitral stenosis (91%).

Age distribution of Rheumatic heart disease and Non Rheumatic causes of atrial fibrillation

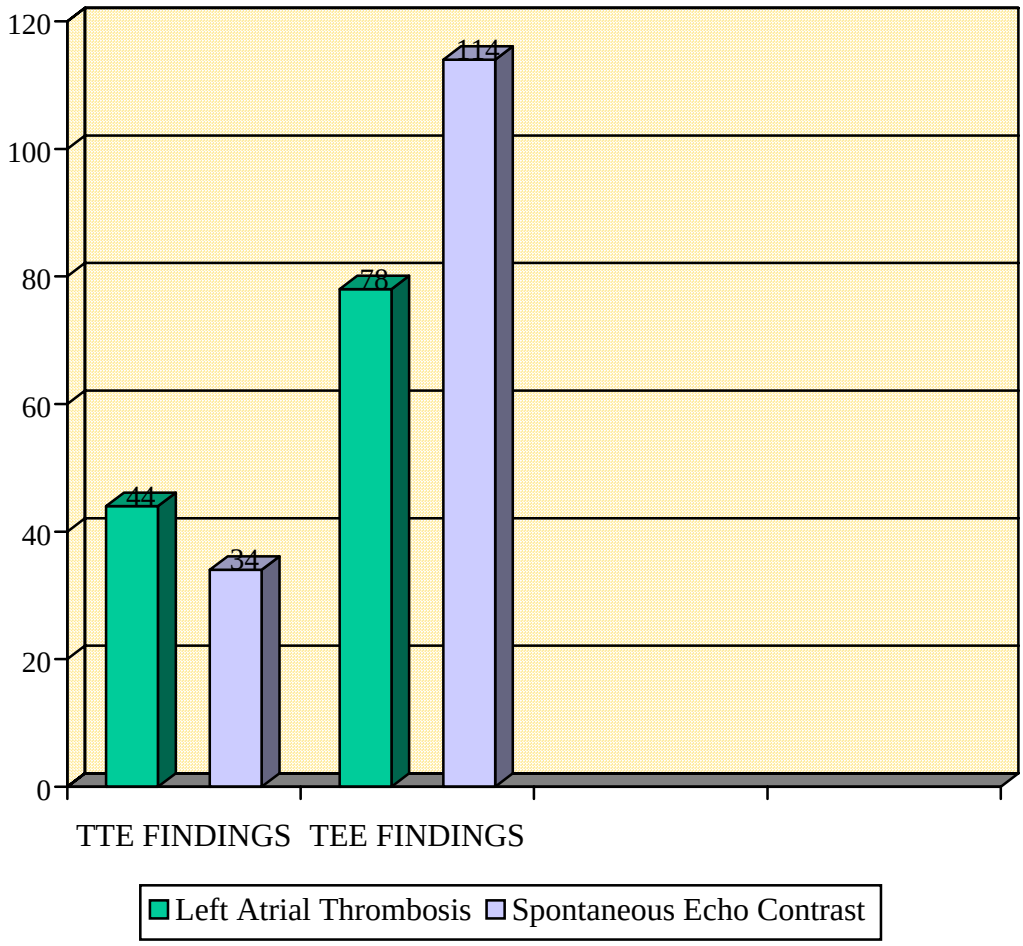
Table 8: Age distribution of Rheumatic and Non Rheumatic Atrial Fibrillation

Age in years	Rheumatic		Non Rheumatic		Total	
	No.	**%**	**No.**	**%**	**No.**	**%**
< 20	14	10.4	-	-	14	9.2
20- 29	32	23.9	-	-	32	21.1
30- 39	52	38.8	2	11.1	54	35.5
40 – 49	32	23.9	10	55.6	42	27.6
50 & above	4	3.0	6	33.3	10	6.6
Total	134	100	18	100	152	100
Mean	47.44		34.13		35.7	
S.D	6.27		8.93		9.7	
'p'	**0.0001(Significant)**					

Rheumatic heart disease was predominant in the middle age group in this study with 116 patients in the age group between 20-50 years. Only 4 patients were present in above 50 years age group. In the Non rheumatic causes group all the patients were above 30 years of age.

Figure 11

Comparison between Transthoracic echocardiography versus Transesophageal echocardiography in Atrial fibrillation



Comparison between Transthoracic echocardiography and Transesophageal echocardiography in Arial fibrillation.

Table 9: Transthoracic Echocardiography versus Transesophageal Echocardiography

Findings	**TTE**		TEE		**'p'**
	No.	**%**	**No.**	**%**	
a) Left atrial Thrombosis	44	28.9	78	51.3	0.0001 Significant
b) Spontaneous Echo Contrast	34	22.4	114	75	0.0001 Significant

Transthoracic echocardiography detected left atrial thrombus in 44 patients (28.9%). Transesophageal echocardiography detected the presence of left atrial thrombus in 78 patients, which constituted around 51.3% of the entire study population. There was a statistically significant difference (p value= 0.0001)

Transthoracic echocardiography detected the presence of spontaneous echo contrast in 34 patients (22.4%), where as Transesophageal echocardiography detected spontaneous echo contrast in 114 (75%). There was a statistically significant difference (p value= 0.0001).

Relationship between mitral valve area and incidence of left atrial thrombus

Table 10: Relationship between Mitral Valve Area and Left Atrial Thrombus in mitral stenosis

Mitral Valve Area (Cm^2)	Thrombus		No Thrombus		Total	
	No.	**%**	**No.**	**%**	**No.**	**%**
0 – 1.0	60	85.7	29	55.8	89	73
1.1 – 1.5	10	14.3	15	28.8	25	20.5
1.6 – 2	-	-	10	15.4	8	6.5
Mean	0.8171		1.05		0.9164	
S.D	0.2213		0.3953		0.3274	
'p'	**0.0138 (Significant)**					

There was a direct association between the mitral valve area and incidence of left atrial thrombus in mitral stenosis. In 89 patients with mitral valve area <1cm^2, left atrial thrombus was present in 60 patients (85.7%). In patients with mitral valve area between 1 to 1.5 cm^2 (22 patients), left atrial thrombus was present in about 10 patients (14.3%). None of the patients with mild mitral stenosis had left atrial thrombus. The difference was statistically significant (p value= 0.0138)

Correlation between mitral valve severity score and incidence of left atrial thrombus in mitral stenosis.

Table 11: Mitral valve Severity Score and Left Atrial Thrombus

Mitral valve Severity Score	**Thrombus**		**No Thrombus**		**Total**	
	No.	**%**	**No.**	**%**	**No.**	**%**
≤ 8	10	14.3	24	46.2	34	27.8
> 8	60	85.7	28	53.8	88	72.2
Mean	7.74		7.08		7.46	
S.D	0.65		1.0		0.88	
'p'	**0.0001 (Significant)**					

34 patients had mitral valve severity score less than 8. In this group 10 patients had left atrial thrombus (14.3% of mitral stenosis patients with LA thrombus). 88 patients with mitral stenosis had mitral valve severity score greater than 8. In this group 60 patients had left atrial thrombus (85.7% of mitral stenosis patients with LA thrombus). The difference was statistically significant (p value=0.0001).

Table 12 :

Relationship between left atrial appendage emptying velocity and left atrial thrombus

Left Atrial Appendage emptying velocity	Thrombus		No Thrombus		Total	
	No.	%	No.	%	No.	%
0.1 - 0.19	62	79.5%	15	20.2	77	50.7
0.2 - 0.29	12	15.4%	10	13.5	22	14.5
0.3 – 0.39	4	5.1%	12	16.2	16	10.5
0.4 and above	-	-	37	51.4	37	24.5
Mean	0.17		0.37		7.46	
S.D	0.O7		0.13		0.88	
'p'	**0.0001 (Significant)**					

77 patients had left atrial appendage emptying velocity between 0.1-0.19 m\sec. In this group of patients 62 had left atrial thrombus and 15 patients did not have thrombus. 22 patients had left atrial emptying velocity between 0.2m\sec – 0.29m\sec, in this group 12 patients had left atrial thrombus. Patients with left atrial emptying velocity between 0.3-0.39m\sec were 16. Out of them 4 had left atrial thrombus. Patients with left atrial appendage emptying velocity greater than 0.4m\sec did not have left atrial appendage thrombus. There was a statistically significant relationship between the left atrial appendage emptying velocity and the incidence of left atrial thrombus, with a p value of 0.0001.

Relationship between left atrial area and incidence of left atrial thrombus

Table 13 Relationship between left atrial size and left atrial thrombus

Left atrial size in cm(2)	Thrombus		No Thrombus		Total	
	No.	%	No.	%	No.	%
2 - 3	5	6.4	41	55.4	46	30.3
3.1 - 4	28	35.9	30	40.5	58	38.2
4.1 – 5	25	32.1	3	4.1	28	18.4
5.1 - 6	17	21.8	-	-	17	11.2
> 6	3	3.8	-	-	3	2
Mean	4.5		2.58		3.76	
S.D	1.0		0.62		1.13	
'p'	**0.0001 (Significant)**					

46 patients had left atrial area in the range of 2-3$cm^{2.}$ In this group only 5 patients had left atrial thrombus and 41 patients did not have any thrombus. 58 patients had left atrial area in the range of 3.1-4 cm^2. In this group 28 patients had left atrial thrombus and 30 patients did not have thrombus. 28 patients had left atrial area in the range of 4.1-5 cm^2. In this group 25 patients had left atrial thrombus and 3 patients did not have left atrial thrombus. All the patients with left atrial area greater than 6 cm^2 had left atrial thrombus. The statistical significance was 0.0001.

Discussion

In our study 152 patients with atrial fibrillation were analyzed. Patients below 18 years of age were excluded. Rheumatic heart disease was the predominant etiology for atrial fibrillation, in this study.

This is in contrast to the western literature where the population studied was predominately whites. The most common causes were systemic hypertension and coronary artery disease in Western countries.

Some Indian studies done previously had shown that Rheumatic heart disease as the commonest cause in our country.

In this study rheumatic heart disease was predominant etiology for atrial fibrillation, 134 (88%) patients had Rheumatic heart disease. Among the Rheumatic heart disease, mitral stenosis was the predominant valvular disease. 122 patients (79%) had Rheumatic mitral stenosis. Hypertensive heart disease was the next common disease. 13 patients (7%) had Hypertensive heart disease. Female patients were predominant in number in our study and 86 patients (57%) were female patients, due to increased prevalence of Rheumatic mitral stenosis in female patients.

But in Framingham study incidence of atrial fibrillation is slightly higher in male population compared to that of females.

Among the patients with rheumatic heart disease with atrial fibrillation, mitral stenosis was the predominant valvular heart disease, 122 patients (79%) had rheumatic mitral stenosis. There was a significant correlation between the incidence of left atrial clot and severity of mitral stenosis. This was well noticed in our study. In our study 89 patients (73%) had severe mitral stenosis. Out of them 68% had left atrial thrombus. We have noticed a marked decline in incidence of left atrial thrombus corresponding to decline in the severity of mitral stenosis. In those with mitral stenosis with moderate severity, 36% ad left atrial thrombus. None of the patients with mitral stenosis of mild severity had left atrial thrombus in our study. In the study by Srimannarayana et al, he found that left atrial clots were present in a third of patients with severe rheumatic mitral stenosis and atrial fibrillation[42]. In our study 68% of patients with severe mitral stenosis had left atrial thrombus.

The incidence of left atrial thrombus in patients with rheumatic heart disease in our study was 54%. In the study by Srimannarayanan et al patients with severe rheumatic mitral stenosis and atrial fibrillation were assessed by Transesophageal echocardiography. Of the 490 patients studied, 163 had left atrial body or left atrial appendage clots. Of the 490 patients who underwent TEE, left atrial clots were present in 163 (32.2%). Isolated left atrial appendage clots were found in 88patients (18%). Isolated left atrial body clots or left atrial appendage clots extending into left atrial body were found in 75 patients (15.3%).[42]

In a small group of 50 patients with mitral stenosis and atrial fibrillation by Hwang et al, the incidence of left atrial thrombus was 56%.[43]

In another small study of 22 patients with mitral stenosis and atrial fibrillation by Karatasaki et al, left atrial thrombus was observed in 12 patients (54%).[44]

These studies point towards the incidence of left atrial thrombus in up to 50% of patients with rheumatic mitral stenosis.

In this study there was a significant relationship between the mitral valve severity score and left atrial thrombus. 34 patients (26.6%) had mitral valve score <8, out of them 10 patients had thrombus (32%). 88 patients (73.3%) had mitral valve score >8, out if them 60 patients (68%) had left atrial thrombus.

In the study by Karatasaki et al, in 22 patents with mitral stenosis and atrial fibrillation, left atrial thrombus was observed in 12 patients (54%). When the severity of mitral valve score is great than 8, the incidence of left atrial clot was about 68% and in patients with mitral valve score less than 8 it was only about 32%. Thus the incidence of left atrial thrombus in patients with Rheumatic mitral stenosis with atrial fibrillation correlates with the mitral valve severity score. The mitral valve severity score takes in to account the mobility, sub valvular thickening, valve thickening and calcification. These patients with mitral valve score >8 are at greater risk of thromboembolism.

In this study Transthoracic echocardiographic examination revealed left atrial clot in 44(28.9%) patients and spontaneous echo contrast was seen in 34 (22.4%)

patients. However Transesophageal echocardiographic examination revealed spontaneous echo contrast in 114 (75%) patients. Left atrial thrombus was seen in 78 (51.3%) patients. Transesophageal echocardiography appears superior in detecting the presence of left atrial thrombus. This was well analyzed in various previous studies. In a study by Manning et al involving 23 patients, 14 patients had left atrial thrombi, 11 of the 14 were confined to the left atrial appendage. At surgery all 12 thrombi identified were confirmed. In this study TEE had a sensitivity of 100% and a specificity of 99% for detection of left atrial thrombi. This study is also complimented by another study by Ashenbeg et al, in which the TEE proved to be superior to TEE in visualizing the left atrial appendage and identifying the left atrial thrombus and with sensitivity that approaches 100%. Thus TEE has got a definite superiority in detecting left atrial thrombus as well as left atrial appendage thrombus as compared with TTE.

In this study population spontaneous echo contrast was seen in 32(21%) patients in transthoracic echocardiography. However Transesophageal echocardiographic examination revealed spontaneous echo contrast in 114(75%) patients. In the study by Fatkins et al Transthoracic echocardiographic studies were performed in 140 patients with atrial fibrillation. Left atrial spontaneous echo contrast was present in 78 patients (56%). Increasing grades of spontaneous echo contrast were associated with decreasing left atrial appendage blood velocity. In multivariate linear regression analysis, the grade of spontaneous echo contrast was significantly and negatively associated with left atrial appendage velocity ($p = 0.001$) and

significantly[46]. Spontaneous echo contrast is the cardiac factor most strongly associated with left atrial appendage thrombus and embolic events.

In Stroke Prevention in Atrial Fibrillation (SPAF III) study, dense spontaneous echo contrast was associated independently with increased thromboembolic risk with a relative risk of 3.7. Because of the frequent association between spontaneous echo contrast and cardio embolic events, a thorough investigation of the left atrial appendage is warranted whenever dense spontaneous echo contrast is identified. Even if thrombus is not found these patients should be considered as high risk for embolic events and treated appropriately.

In this study Transesophageal echocardiography proved to be far better in eliciting the risk factors for thromboembolic in patients with atrial fibrillation, when compared to that of transthoracic echocardiography. In the BEST trial comparing Transesophageal echocardiography to surgical findings, sensitivity of Transesophageal echocardiography to left atrial thrombus was 100% and specificity of 99%. Transesophageal echocardiography has a higher sensitivity than transthoracic echocardiography for spontaneous echo contrast. Spontaneous echo contrast was detected by Transesophageal echocardiography in 25 – 45% of atrial fibrillation patients and in >80% of those with atrial fibrillation and left atrial thrombus[47].

Transesophageal echo offers unique imaging resolution of the left atrium and its appendage and is an excellent tool for detecting embolic sources. Transesophageal Echocardiographic features that are independently associated with increased

thromboembolic risk are appendage thrombi, dense spontaneous echo contrast, left atrial appendage peak velocity <20 cm\sec. In this study patient with left atrial appendage emptying velocity less than 0.2m\sec had increased incidence of left atrial thrombus. This study correlates well with the study conducted by Fatkins et al .[46]

In this study there was a strong correlation between size of left atrium and the incidence of left atrial thrombus. The mean size of the left atrium was 4.5 cm^2 in patients who had left atrial thrombus; where as the mean left atrial size was around 2.5 cm^2 in patients with out thrombus.

CONCLUSION

1. Rheumatic heart disease was the most common cause of atrial fibrillation in this study.
2. Rheumatic mitral stenosis was the most common valvular lesion associated with atrial fibrillation.
3. Transesophageal echocardiography was superior to transthoracic echocardiography, regarding the evaluation of atrial fibrillation, because of its better sensitivity to left atrial thrombus as well as spontaneous echo contrast.
4. In Rheumatic mitral stenosis mitral valve severity score correlates with the incidence of left atrial thrombus. Mitral valve score >8 had thrombus had thrombus in 68% where as left atrial thrombus was present in 26.6% of patients with mitral valve score <8.
5. In Rheumatic mitral stenosis, valve area also correlated with the incidence of left atrial thrombus. 73% of patients with severe mitral stenosis had left atrial thrombus; where as none of the patients with mild mitral stenosis had left atrial thrombus.
6. Patients with left atrial appendage emptying velocity <0.2m/sec had increased incidence of left atrial thrombus in this study.
7. Patients with left atrial size > 4.5 cm^2 had increased incidence of thrombus formation in the left atrium in patients with atrial fibrillation in this study.

SUMMARY

Atrial fibrillation is the most common sustained cardiac arrhythmia and represents the most common clinically significant cardiac arrhythmia. It is the most common cause of embolisation from cardiac source leading to cerebrovascular accident. The study was undertaken to study the clinical profile, under lying causes of atrial fibrillation and also to do a comparative analysis of findings in Transthoracic and Transesophageal echocardiography in patients with atrial fibrillation.

Thus 152 patients with atrial fibrillation were taken up for study, after excluding the patients with age <18 years of age. After institutional ethical clearance, and an informed consent, the patient's sociodemographic, clinical and laboratory data were collected and analyzed statistically.

Mean age of study population was 35.7±10 years. Female patients were predominant in our study. 86 patients (56.6%) were female patients.

Predominant number of patients in our study group belonged to rheumatic heart disease, with 134 patients (88%) had rheumatic heart disease and 18 patients (12%) belonged to non rheumatic group. Systemic hypertension was second most common cause.

In rheumatic heart disease group 122 patients (122\152) had mitral stenosis. Out of those with mitral stenosis, 89 patients had severe mitral stenosis 25 patients had moderate mitral stenosis and 8 patients had mild mitral stenosis.

Thus rheumatic mitral stenosis was the most common cause of atrial fibrillation in this study with slight female predilection.

The incidence of left atrial thrombus had a direct correlation to the severity of the mitral stenosis. In this study 89 patients had severe mitral stenosis and out of them 68% had left atrial thrombus, where as in patients with moderately severe mitral stenosis only 36% had left atrial thrombus and in patients with mild mitral stenosis none had left atrial thrombus.

In this study, patients with mitral valve severity <8 were about 34, out of them 10 patients (26%) had left atrial thrombus. 88 patients (72.2%) had mitral valve severity score >8 and out of them 60 patients (68%) had left atrial thrombus. Thus severity of mitral stenosis and mitral valve score correlated with the incidence of left atrial clot in patients with mitral stenosis.

In this study patients underwent transthoracic echocardiographic evaluation followed by Transesophageal echocardiographic evaluation.

Transthoracic echocardiography detected left atrial thrombus in 44 patients (28.9%), where as Transesophageal echocardiography showed left atrial thrombus in 78 patients (51.3%).

Like wise, Transthoracic echocardiography detected spontaneous echo contrast in 34 patients (22.4%). Transesophageal echocardiography revealed spontaneous echo contrast in 114 patients (75%).

Thus in this study Transesophageal echocardiography proved to be superior to Transthoracic echocardiography, regarding the detection of left atrial thrombus as well as detection of spontaneous echo contrast.

Patients with left atrial appendage flow velocity in range of 0.2m\sec had increased risk of thrombus formation

Patients with left atrial size around 4.5 cm^2 had statistically significant risk of thrombus formation.

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PROFORMA

ATRIAL FIBRILLATION; CLINICAL, TRATHORACIC

ECHOCARDIOGRAPHY AND TRANSESOPHAGEAL

ECHOCARDIOPATHY CORRELATION

NAME	C.D. NO	
AGE	I.P. NO.	
SEX	WARD NO.	DOA
OCCUPATION	UNIT	DOD

DIAGNOSIS :

CAUSE OF ATRIAL FIBRILLATION :

HISTORY :

Risk Factors :

Age > 65 Yrs CAD CHF COPD DM

Previous History of TIA / Stroke

Significant Valvular Heart Disease

Hypertension

General Examination :

Heart Rate :	B.P	BUILT
Pulse Rate :	Tremor	Eye Signs
Pulse Deficit		

Cardiovascular System : JVP

Mitral Area

Tricuspid Area

Aortic Area

Pulmonary Area

Respiratory System :

Per Abdomen :

Central Nervous System :

Investigations

ECG Rate Rhythm P-R Interval QRS Duration

P-Axis QRS Axis ST-T Changes LAE RAE LVH RVH

CXR

Trans Thoracic Echo :

LVIDd LVIDs EF

IVSs IVSd

LVPWs LVPWd

RWMA

DOPPLER

MV E A E/A DT

IVRT

TRANS ESOPHAGEAL ECHOCARDIOGRAPHY

LA Size

LV functional status

Spontaneous Echo Contrast

LA appendage emptying velocity

Complex Aortic Atheroma

LA clot

LVID - Left ventricular internal Diameter
d- diastolic
s- systolic

IVS - Inter ventricular septum
d- diastolic
s- systolic

EF - Ejection Fraction

LVPW - Left ventricular posterior Wall
d- diastolic
s- systolic

RWMA - Regional Wall Motion Abnormality

MV - Mitral Valve

IVRT - Iso volumetric relaxation time

MASTER CHART

S.NO	Age	SEX	CAD	RHD	HT	CM	CHD	THYROID	CVA	TTE									TEE						
													MS												
										LA Size (cm^2)	LA CLOT	AUTO CONTRAST	Thickness of Mitral Valve	Mobility of mitral valve	SVA	Calcium	Severety Score	Mitral orifice in cm	MR	AR	AS	LAA Size	LAA Emptying velocity	LA THROMBUS	Clot size
1	34	2	-	+	-	-	-	-	-	3.3	+	+	2+	2+	2+	2+	8	0.9	II	-	-	3.6	0.1	1	1.1
2	18	1	-	+	-	-	-	-	-	5.9	-	-	2+	2+	2+	-	6	1.3	II	1	-	3.2	0.23	1	0.91
3	47	1	-	-	-	-	-	+	-	2.5	-	-										2	0.4	2	
4	19	2	-	+	-	-	-	-	-	3.1	-	-	2+	2+	2+	2+	8	0.7	-	-	-	2.7	0.4	2	
5	36	1	-	+	-	-	-	-	-	3.2	-	-	2+	2+	2+	-	6	1.5	II	II	-	3.9	0.5	2	
6	28	1	-	+	-	-	-	-	-	4.7	-	-	2+	2+	2+	-	6	1.3	II	-	-	3.1	0.12	1	0.42
7	27	2	-	+	-	-	-	-	+	4	+	-	2+	2+	2+	2+	8	0.8	I	-	-	3.9	0.12	1	6.93
8	54	2	-	-	-	-	+	-	-	2.1	-	-										3.1	0.15	2	
9	32	2	-	+	-	-	-	-	-	4	+	-	2+	2+	2+	2+	8	0.7	I	II	-	3.2	0.19	1	0.72
10	34	1	-	+	-	-	-	-	-	3.2	+	-	2+	2+	2+	-	6	1.2	I	-	-	3.4	0.2	1	1.4
11	56	2	-	+	-	-	-	-	-	4.6	-	-	2+	2+	2+	2+	8	0.8	I	-	-	3.7	0.25	1	1.8
12	18	2	-	+	-	-	-	-	-	4	-	-	2+	2+	2+	-	6	1.6	II	-	-	3.4	0.4	2	
13	32	1	-	+	-	-	-	-	-	5	-	-	2+	2+	2+	2+	8	0.7	I	-	-	3.2	0.4	2	
14	26	1	-	+	-	-	-	-	-	4.5	-	-	2+	2+	2+	2+	8	0.9	-	-	II	3.6	0.19	1	1.19
15	34	2	-	+	-	-	-	-	+	4	-	-	2+	2+	2+	2+	8	0.7	I	II	-	2.1	0.13	1	7.13
16	28	2	-	+	-	-	-	-	-	4	-	-	2+	2+	2+	-	6	1.3	II	II	-	2.4	0.4	2	
17	56	1	-	-	-	-	-	+	-	2	-	-										2	0.4	2	
18	31	1	-	+	-	-	-	-	-	4.4	-	-							II	II	-	2.3	0.3	2	
19	28	1	-	+	+	-	-	-	-	4	+	+	2+	2+	2+	2+	8	0.8	I	I	-	3.5	0.12	1	0.7
20	42	1	-	+	+	-	-	-	-	5	+	+	2+	2+	2+	2+	8	0.6	-	-	-	5	0.12	1	0.42
21	26	2	-	+	-	-	-	-	+	5.3	+	-	2+	2+	2+	2+	8	0.7	I	II	II	5.7	0.1	1	1.87
22	33	2	-	+	-	-	-	-	-	4	-	-	2+	2+	2+	-	6	1.6	II	-	-	2	0.4	2	
23	27	1	-	+	-	-	-	-	-	5	+	-	2+	2+	2+	2+	8	0.8	-	-	-	3.4	0.1	1	0.11
24	46	2	-	-	+	-	-	-	-	3.2	-	-										4	0.4	2	
25	26	2	-	+	-	-	-	-	+	5	-	+	2+	2+	2+	2+	8	0.6	-	-	-	3.1	0.27	1	0.84
26	48	2	-	-	+	-	-	-	-	2.5	-	-										3.2	0.4	2	
27	46	2	-	+	-	-	-	-	-	5.9	-	-	2+	2+	2+	2+	8	0.7	II	-	-	3.6	0.1	1	1.32
28	18	1	-	+	-	-	-	-	-	4.5	-	-							II	-	-	3.6	0.4	2	
29	36	2	-	+	-	-	-	-	-	3.5	-	-							II	I	-	3.1	0.5	2	
30	32	1	-	+	-	-	-	-	-	5	+	+	2+	2+	2+	2+	8	0.8	II	-	-	5.9	0.15	1	1.32
31	34	1	+	-	-	+	-	-	-	2.4	-	-										3.1	0.24	1	1.1

S.NO	Age	SEX	CAD	RHD	HT	CM	CHD	THYROID	CVA	TTE										TEE						
													MS													
										LA Size (cm^2)	LA CLOT	AUTO CONTRAST	Thickness of Mitral Valve	Mobility of mitral valve	SVA	Calcium	Severety Score	Mitral orifice in cm	MR	AR	AS	LAA Size	LAA Emptying velocity	LA THROMBUS	Clot size
32	19	2	-	+	-	-	-	-	-	5.9	-	-	2+	2+	2+		6	1.7	-	I	-	5.9	0.5	2	
33	38	2	-	+	-	-	-	-	-	5.8	-	+	2+	2+	2+	1	7	1.5	-	I	-	5.9	0.13	1	0.63
34	46	2	-	-	-		-	-	+	2.3	-	-										3	0.15	1	7.82
35	41	2	-	+	-	-	-	-	-	5	-	-	2+	2+	2+	2+	8	0.8	I	-	-	2.1	0.15	1	0.48
36	36	1	-	+	-	-	-	-	-	6.8	+	-	2+	2+	2+	2+	8	0.6	-	-	-	3	0.15	1	1.12
37	44	1	-	+	-	-	-	-	-	5.6	-	-							I	-	-	4.4	0.16	1	1.17
38	36	2	-	+	-	-	-	-	-	3.8	-	-	2+	2+	2+	2+	8	0.8	-	-	-	4	0.12	1	0.8
39	52	1	-	-	-	+	-	-	-	2.6	-	-										2.1	0.4	2	
40	19	2	-	+	-	-	-	-	-	3.5	-	-	2+	2+	2+	-	6	1.2	I	-	-	2	0.2	2	
41	45	2	-	+	-	-	-	-	-	3.6	-	+	2+	2+	2+	1	6	1.7	-	-	-	2	0.3	2	
42	48	1	-	+	-	-	-	-	-	3.5	-	-							-	-	-	2	0.4	2	
43	44	1	-	+	-	-	-	-	-	4.5	-	-	2+	2+	2+	2+	8	0.7	-	-	-	2	0.3	2	
44	33	1	-	+	-	-	-	-	-	3.8	+	-	2+	2+	2+	-	6	1.2	-	-	-	4	0.15	1	0.36
45	36	2	-	+	-	-	-	-	-	4.8	+	-	2+	2+	2+	2+	8	0.7	-	-	-	4	0.17	2	
46	37	1	-	+	-	-	-	-	-	1.5	+	-	2+	2+	2+	2+	8	0.6	-	II	-	4	0.1	1	0.52
47	42	2	-	+	-	-	-	-	-	5	-	-	2+	2+	2+	2+	8	0.6	-	-	-	2	0.4	2	
48	47	1	-	-	-	-	-	+	-	4.4	-	-										2.3	0.3	2	
49	34	2	-	+	-	-	-	-	-	5	+	-	2+	2+	2+	2+	8	0.7	-	-	-	4	0.15	1	1.12
50	31	2	-	+	-	-	-	-	-	5	-	-							-	-	-	4	0.16	1	1.17
51	36	1	-	+	-	-	-	-	-	5	-	-	2+	2+	2+	2+	8	0.8	II	-	-	4	0.1	1	1.3
52	38	2	-	+	-	-	-	-	-	4	-	+	2+	2+	2+	2+	8	0.7	-	-	-	2	0.38	2	
53	49	2	-	+	-	-	-	-	-	4	-	+	2+	2+	2+	2+	8	0.6	-	-	-	2	0.4	2	
54	47	2	-	+	-	-	-	-	-	3	-	+	2+	2+	2+	2+	8	0.9	-	-	-	4	0.13	1	1.68
55	45	2	-	+	-	-	-	-	-	5.4	-	-	2+	2+	2+	2+	8	0.7	II	-	-	1.8	0.38	2	
56	44	1	-	+	-	-	-	-	-	3.8	-	+	2+	2+	2+	2+	8	0.8	II	-	-	4	0.18	1	1.43
57	28	2	-	+	-	-	-	-	-	3	-	-	2+	2+	2+	2+	8	0.7	-	-	-	1.4	0.13	1	0.84
58	42	2	-	+	-	-	-	-	-	3	-	-							II	II	-	1.6	0.4	2	
59	46	1	-	+	-	-	-	-	-	4	+	+	2+	2+	2+	2+	8	0.9	-	II	-	1.8	0.4	2	
60	29	1	-	+	-	-	-	-	-	4.8	-	-	2+	2+	2+	2+	8	0.8	II	-	-	3.6	0.14	1	1.12
61	27	2	-	+	-	-	-	-	-	3.5	-	-	2+	2+	2+	2+	8	0.7	II	-	-	1.8	0.4	2	
62	32	2	-	+	-	-	-	-	-	3.8	-	+	2+	2+	2+	2+	8	0.8	-	-	-	1.8	0.3	2	
63	36	2	-	+	-	-	-	-	-	4	-	+	2+	2+	2+	2+	8	0.7	-	-	-	2	0.18	2	
64	44	2	-	+	-	-	-	-	-	5	-	-	2+	2+	2+	2+	8	0.6	-	-	-	4	0.18	1	1.54
65	54	2	-	-	-	-	+	-	-	5.4	-	-										1.8	0.38	2	
66	36	2	-	+	-	-	-	-	-	4	+	-	2+	2+	2+	2+	8	0.8	I	II	-	3.2	0.17	1	0.7

S.NO	Age	SEX	CAD	RHD	HT	CM	CHD	THYROID	CVA	TTE										TEE						
													MS													
										LA Size (cm^2)	LA CLOT	AUTO CONTRAST	Thickness of Mitral Valve	Mobility of mitral valve	SVA	Calcium	Severety Score	Mitral orifice in cm	MR	AR	AS	LAA Size	LAA Emptying velocity	LA THROMBUS	Clot size
67	38	1	-	+	-	-	-	-	-	3.2	+	-	2+	2+	2+	-	6	1.4	I	-	-	3.4	0.2	2	
68	55	2	-	+	-	-	-	-	-	6.6	-	-	2+	2+	2+	2+	8	0.7	I	-	-	3.7	0.4	2	
69	44	2	-	-	+	-	-	-	-	4	-	-	2+	2+	2+	-	6	1.3	-	-	-	3.2	0.3	2	
70	19	2	-	+	-	-	-	-	-	6	-	-	2+	2+	2+	-	6	1.2	III	-	-	3.4	0.4	2	
71	32	1	-	+	-	-	-	-	-	5	-	-	2+	2+	2+	2+	8	0.7	I	-	-	3.2	0.4	2	
72	27	1	-	+	-	-	-	-	-	6	-	-	2+	2+	2+	-	6	1.4	III	-	-	3.4	0.4	2	
73	56	1	-	-	-	-	-	+	-	5	-	-										3.4	0.4	2	
74	29	1	-	+	+	-	-	-	-	4	+	+	2+	2+	2+	2+	8	0.7	I	I	-	3.5	0.12	1	0.84
75	44	1	-	+	+	-	-	-	-	5	+	+	2+	2+	2+	2+	8	0.6	-	-	-	5	0.13	1	1.28
76	28	2	-	+	-	-	-	-	-	5.3	+	-	2+	2+	2+	2+	8	0.8	I	II	II	5.7	0.1	1	1.12
77	32	2	-	+	-	-	-	-	-	4	-	-	2+	2+	2+	-	6	1.5	II	-	-	2	0.4	2	
78	26	1	-	+	-	-	-	-	-	5	+	-	2+	2+	2+	2+	8	0.7	-	-	-	3.4	0.1	1	1.87
79	28	1	-	+	-	-	-	-	-	5	+	+	2+	2+	2+	2+	8	0.6	I	-	-	3.8	0.19	1	0.77
80	34	2	-	+	-	-	-	-	-	4	+	+	2+	2+	2+	2+	8	0.9	II	-	-	3.5	0.12	1	1.1
81	18	1	-	+	-	-	-	-	-	4.6	-	-	2+	2+	2+	-	6	1.3	II	1	-	3.7	0.2	1	0.91
82	19	2	-	+	-	-	-	-	-	5.9	-	-	2+	2+	2+	2+	8	0.7	-	-	-	3.6	0.1	2	
83	36	1	-	+	-	-	-	-	-	3.8	-	-	2+	2+	2+	-	6	1.5	II	II	-	4	0.2	2	
84	46	2	-	-	+	-	-	-	-	2.7	-	-										3	0.15	2	
85	28	1	-	+	-	-	-	-	-	1.5	-	-	2+	2+	2+	-	6	1.3	II	-	-	4	0.1	1	0.42
86	27	2	-	+	-	-	-	-	+	4	+	-	2+	2+	2+	2+	8	0.8	I	-	-	3.9	0.2	1	6.93
87	32	2	-	+	-	-	-	-	-	5.9	+	-	2+	2+	2+	2+	8	0.7	I	II	-	3.2	0.23	1	0.72
88	34	1	-	+	-	-	-	-	-	4	+	-	2+	2+	2+	-	6	1.2	I	-	-	2	0.18	1	1.4
89	56	2	-	+	-	-	-	-	-	3.5	-	-	2+	2+	2+	2+	8	0.8	I	-	-	2	0.14	1	1.8
90	18	2	-	+	-	-	-	-	-	5	-	-	2+	2+	2+	-	6	1.6	II	-	-	5	0.2	2	
91	32	1	-	+	-	-	-	-	-	3.5	-	-	2+	2+	2+	2+	8	0.7	I	-	-	2	0.2	2	
92	48	2	-	-	+	-	-	-	-	5	-	-										3.2	0.4	2	
93	26	1	-	+	-	-	-	-	-	3.3	-	-	2+	2+	2+	2+	8	0.9	-	-	II	3.6	0.16	1	1.19
94	34	2	-	+	-	-	-	-	+	4	-	-	2+	2+	2+	2+	8	0.7	I	II	-	2	0.17	1	7.13
95	28	2	-	+	-	-	-	-	-	5	-	-	2+	2+	2+	-	6	1.3	II	II	-	3.4	0.1	2	
96	31	1	-	+	-	-	-	-	-	2.2	-	-							II	II	-	3.1	0.15	2	
97	28	1	-	+	+	-	-	-	-	4	+	+	2+	2+	2+	2+	8	0.8	I	I	-	2	0.18	1	0.7
98	42	1	-	+	+	-	-	-	-	4.8	+	+	2+	2+	2+	2+	8	0.6	-	-	-	4	0.17	1	0.42
99	26	2	-	+	-	-	-	-	+	5	+	-	2+	2+	2+	2+	8	0.7	I	II	II	3.1	0.2	1	1.87
100	33	2	-	+	-	-	-	-	-	4.7	-	-	2+	2+	2+	-	6	1.6	II	-	-	3.1	0.12	2	
101	52	1	-	-	-	+	-	-	-	3	-	-										1.6	0.4	2	

S.NO	Age	SEX	CAD	RHD	HT	CM	CHD	THYROID	CVA	TTE										TEE						
													MS													
										LA Size (cm^2)	LA CLOT	AUTO CONTRAST	Thickness of Mitral Valve	Mobility of mitral valve	SVA	Calcium	Severety Score	Mitral orifice in cm	MR	AR	AS	LAA Size	LAA Emptying velocity	LA THROMBUS	Clot size
102	27	1	-	+	-	-	-	-	-	4.5	+	-	2+	2+	2+	2+	8	0.8	-	-	-	3.6	0.19	1	0.11
103	26	2	-	+	-	-	-	-	+	5	-	+	2+	2+	2+	2+	8	0.6	-	-	-	4	0.15	1	0.84
104	46	2	-	+	-	-	-	-	-	4	-	-	2+	2+	2+	2+	8	0.7	II	-	-	3.2	0.2	1	1.32
105	18	1	-	+	-	-	-	-	-	2.3	-	-							II	-	-	4	0.4	2	
106	34	1	+	-	-	+	-	-	-	5.3	-	-										5.7	0.1	1	1.1
107	36	2	-	+	-	-	-	-	-	3.5	-	-							II	I	-	1.8	0.4	2	
108	32	1	-	+	-	-	-	-	-	4	+	+	2+	2+	2+	2+	8	0.8	II	-	-	2	0.14	1	1.32
109	19	2	-	+	-	-	-	-	-	3	-	-	2+	2+	2+		6	1.7	-	I	-	1.4	0.3	2	
110	38	2	-	+	-	-	-	-	-	4.5	-	+	2+	2+	2+	1	7	1.5	-	I	-	3.6	0.19	1	0.63
111	41	2	-	+	-	-	-	-	-	3.1	-	-	2+	2+	2+	2+	8	0.8	I	-	-	2.7	0.16	1	0.48
112	36	1	-	+	-	-	-	-	-	5	+	-	2+	2+	2+	2+	8	0.6	-	-	-	3.4	0.1	1	1.12
113	44	1	-	+	-	-	-	-	-	3.8	-	-							I	-	-	4	0.15	1	1.17
114	36	2	-	+	-	-	-	-	-	3.8	-	-	2+	2+	2+	2+	8	0.8	-	-	-	1.8	0.13	1	0.8
115	19	2	-	+	-	-	-	-	-	2.7	-	-	2+	2+	2+	-	6	1.2	I	-	-	2	0.4	2	
116	45	2	-	+	-	-	-	-	-	4	-	+	2+	2+	2+	1	6	1.7	-	-	-	1.8	0.4	2	
117	48	1	-	+	-	-	-	-	-	3.6	-	-							-	-	-	2	0.3	2	
118	44	1	-	+	-	-	-	-	-	5	-	-	2+	2+	2+	2+	8	0.7	-	-	-	5	0.2	2	
119	33	1	-	+	-	-	-	-	-	3.2	+	-	2+	2+	2+	-	6	1.2	-	-	-	3.9	0.17	1	0.36
120	36	2	-	+	-	-	-	-	-	3.5	+	-	2+	2+	2+	2+	8	0.7	-	-	-	3.1	0.5	2	
121	37	1	-	+	-	-	-	-	-	5	+	-	2+	2+	2+	2+	8	0.6	-	II	-	2	0.12	1	0.52
122	42	2	-	+	-	-	-	-	-	5	-	-	2+	2+	2+	2+	8	0.6	-	-	-	4	0.18	2	
123	34	2	-	+	-	-	-	-	-	2.1	+	-	2+	2+	2+	2+	8	0.7	-	-	-	2	0.11	1	1.12
124	31	2	-	+	-	-	-	-	-	3.5	-	-							-	-	-	2.1	0.1	1	1.17
125	46	2	-	-	-		-	-	+	5	-	-										2.1	0.25	1	7.82
126	36	1	-	+	-	-	-	-	-	6.6	-	-	2+	2+	2+	2+	8	0.8	II	-	-	3.7	0.17	1	1.3
127	38	2	-	+	-	-	-	-	-	5	-	+	2+	2+	2+	2+	8	0.7	-	-	-	4	0.1	2	
128	49	2	-	+	-	-	-	-	-	5.9	-	+	2+	2+	2+	2+	8	0.6	-	-	-	5.9	0.5	2	
129	47	2	-	+	-	-	-	-	-	4	-	+	2+	2+	2+	2+	8	0.9	-	-	-	3.4	0.13	1	1.68
130	45	2	-	+	-	-	-	-	-	4	-	-	2+	2+	2+	2+	8	0.7	II	-	-	3.2	0.29	2	
131	44	1	-	+	-	-	-	-	-	4.5	-	+	2+	2+	2+	2+	8	0.8	II	-	-	2	0.3	1	1.43
132	28	2	-	+	-	-	-	-	-	4.8	-	-	2+	2+	2+	2+	8	0.7	-	-	-	3.6	0.14	1	0.84
133	42	2	-	+	-	-	-	-	-	5	-	-							II	II	-	3.8	0.2	2	
134	46	1	-	+	-	-	-	-	-	5.3	+	+	2+	2+	2+	2+	8	0.9	-	II	-	5.7	0.1	2	
135	29	1	-	+	-	-	-	-	-	5.6	-	-	2+	2+	2+	2+	8	0.8	II	-	-	4.4	0.26	1	1.12
136	27	2	-	+	-	-	-	-	-	4	-	-	2+	2+	2+	2+	8	0.7	II	-	-	2.4	0.4	2	

S.NO	Age	SEX	CAD	RHD	HT	CM	CHD	THYROID	CVA	TTE										TEE						
													MS													
										LA Size (cm^2)	LA CLOT	AUTO CONTRAST	Thickness of Mitral Valve	Mobility of mitral valve	SVA	Calcium	Severety Score	Mitral orifice in cm	MR	AR	AS	LAA Size	LAA Emptying velocity	LA THROMBUS	Clot size
137	32	2	-	+	-	-	-	-	-	3.2	-	+	2+	2+	2+	2+	8	0.8	-	-	-	3.4	0.2	2	
138	36	2	-	+	-	-	-	-	-	5	-	+	2+	2+	2+	2+	8	0.7	-	-	-	4	0.2	2	
139	44	2	-	+	-	-	-	-	-	3.2	-	-	2+	2+	2+	2+	8	0.6	-	-	-	3.4	0.2	1	1.54
140	36	2	-	+	-	-	-	-	-	2.8	+	-	2+	2+	2+	2+	8	0.8	I	II	-	3.2	0.0.2	1	0.7
141	38	1	-	+	-	-	-	-	-	4	+	-	2+	2+	2+	-	6	1.4	I	-	-	3.5	0.12	2	
142	55	2	-	+	-	-	-	-	-	3.8	-	-	2+	2+	2+	2+	8	0.7	I	-	-	4	0.18	2	
143	44	2	-	-	+	-	-	-	-	6.8	-	-	2+	2+	2+	-	6	1.3	-	-	-	3	0.15	2	
144	19	2	-	+	-	-	-	-	-	4	-	-	2+	2+	2+	-	6	1.2	III	-	-	2.1	0.3	2	
145	32	1	-	+	-	-	-	-	-	5	-	-	2+	2+	2+	2+	8	0.7	I	-	-	3.2	0.4	2	
146	27	1	-	+	-	-	-	-	-	5.8	-	-	2+	2+	2+	-	6	1.4	III	-	-	5.9	0.13	2	
147	29	1	-	+	-	-	-	-	-	4	+	+	2+	2+	2+	2+	8	0.7	I	I	-	2	0.28	1	0.84
148	44	1	-	+	+	-	-	-	-	4	+	+	2+	2+	2+	2+	8	0.6	-	-	-	3.2	0.22	1	1.28
149	28	2	-	+	-	-	-	-	-	2.6	+	-	2+	2+	2+	2+	8	0.8	I	II	II	3.1	0.11	1	1.12
150	32	2	-	+	-	-	-	-	-	6	-	-	2+	2+	2+	-	6	1.5	II	-	-	3.4	0.4	2	
151	26	1	-	+	-	-	-	-	-	3	+	-	2+	2+	2+	2+	8	0.7	-	-	-	4	0.13	1	1.87
152	28	1	-	+	-	-	-	-	-	5	+	+	2+	2+	2+	2+	8	0.6	I	-	-	5.9	0.15	1	0.77

Sex - 1 Male: 2 Female
CAD - Coronary Artery Disease
RHD - Rheumatic Heart Disease
HT - Hyper Tension
CM - Cardio Myopathy
CHT - Congenital Heart Disease
CVA - Cerebro Vascular Accident
TTE - Trans Thoracic Echo Cardiography
TEE - Trans Esophageal Echo Cardiography
SVA - Sub-Vavular Apparatus
MS - Mitral Stenosis
MR - Mitral Regurgitation
AR - Aortic Regurgitation
AS - Aortic Stenosis
LAA - Left Atrial Appendage
SEC - Spontaneous Echo Contrast